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Anaesthesia for Repair of RupturedMeningomyelocoele in a Neonate: A Case Report

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Summary: DT was a 22-day-old female neonate who had repair of a ruptured meningomyelocoele under general anaesthesia with muscle relaxation and controlled ventilation. Anaesthesia was maintained with a balanced technique for neonates. Standard anaesthetic monitoring was performed. She was manually ventilated with 100% oxygen using Mapleson F breathing system. Hypothermia was prevented by increasing ambient temperature, warm infusion and use of over-head radiant heater. Surgery was done with the patient in prone position. She had uneventful postoperative recovery and was discharged home on 20th post-operative day. The perioperative challenges in this case are discussed.

Key Words:Anaesthesia, neonate, meningomyelocoele, repair, surgery.

INTRODUCTION

Meningomyelocoele is the protrusion of a part of the meninges and spinal cord through a defect in the vertebral column. It is the most complex congenital malformation of the central nervous system that is compatible with life.[1] It is also a common neurosurgical abnormality and is often associated with other central nervous system abnormalities such as talipes, paraparesis and congenital hydrocephalus.[2] The incidence varies from 0.1 to 1% of live births.[3] There is a familial tendency and the risk of recurrence after one child is about 5%.[3,4]

Failure of fusion of vertebral arches during embryogenesis gives rise to spinal bifida which is frequently associated with maldevelopment of the spinal cord and membranes.[4] Rupture of meningomyelocoele could be fatal because of the possibility of brain stem herniation especially when associated with hydrocephalus.[5] In addition, central nervous system infection due to absence of normal protective covering of the spinal cord can occur, thus early repair of meningomyelocoelewithin the 1st week of life is indicated. The procedure involves a careful reconstruction of the dura and over lying tissues with the patient in the prone position.

The anaesthetic management of a newborn for surgery requires consideration of the surgical lesion and its systemic effects, the potential for operative blood loss and special requirements such as the patient's position in relation to monitoring devices and vascular catheters.[6,7] In this patient however, the main anaesthetic challenges were limited to the evaporative fluid loss from meningomyelocoele, predisposition to the infection of the central nervous system from the patulous anus and the difficulty of placing the patient in the supine position during intubation and managing the neonate in the prone position.The incidence of serious anaesthetic complications is significantly higher in neonates than in older children and adults. Provision of safe anaesthesia is better assured when specialized equipment and techniques necessary to conduct anaesthesia are performed by experienced anaesthetist who understand the anatomic, physiologic and pathologic differences that characterize these patients during their change from intrauterine life to extrauterine life.

Preoperative preparation aims to stabilize the cardiopulmonary system, body temperature and metabolic functions and, provide energy substrate to meet immediate metabolic needs. Meticulous attention to all aspects of anaesthesia and the surgical procedure combined with the use of appropriate monitoring devices will greatly enhance the safety of anaesthesia in neonates.

CASE PRESENTATION

DT is a 22-day-old female neonate who was admitted into the special care baby unit (SCBU) of the University of Port Harcourt Teaching Hospital, Rivers State, Nigeria with a history of non-tender cystic swelling in the sacral region which was not increasing in size. The cystic mass had ruptured on the 2nd day of life.She was delivered by spontaneous vaginal delivery at 37 weeks of gestation to a 26-year-old P2+0 teacher in a maternity home following prolonged rupture of membrane. She weighed was 3.1 kg at birth and had fever and tinge of jaundice which was noticed on the first day of life. Her mother received routine antenatal care in a government approved maternity home and she had taken only the prescribed drugs during pregnancy.

Physical examination revealed an active neonate with normal head circumference, normotensive fontanels and cystic swelling on the sacral region,she was neither pale, cyanosed nor in any respiratory distress. However, she was jaundiced, dehydrated and febrile. The lesion over the lumbosacral area measured about 5 cm by 6 cm. There was an ulcer over the lower aspect of the lesion which was firm and discharging purulent fluid. The heart rate of 152 beats/minute, and heart sounds were normal with no mumur. The respiratory rate was 50 breaths per minute and breath sounds were normal. The baby was alert. There was reduced tone in both lower limbs. The neonate was noticedwith faecal and urinary incontinence. Other systems were essentially normal. A diagnosis of neonatal sepsis and jaundice in a child with meningomyelocoele was made.

She continued breast feeding 3-hourly while 10% dextrose saline 80 mls/kg/day using soluset was infused. Sepsis was controlled with intravenous ceftriaxone 150 mg daily and

gentamycin 7.5 mg 12 hourly. The defect was cleaned and packed with saline –soaked gauze. She also received phototherapy.Two days after presentation, the patient developed fullness of the anterior fontanelle. A diagnosis of early meningitis was made and mannitol 1 gm/kg over 1 hour, 8-hourly for 24 hours administered. 1ntravenous ceftriaxone was increased to 200 mg daily, while gentamycin 7.5 mg was administered 8-hourly.

She was exposed to phototherapy and series of bilirubin estimation were carried out. The serum electrolyte result revealed acidosis and it was corrected with administration of intravenous sodium bicarbonate 2mls/kg in double dilution 8 hourly for 24 hours. She was reviewed by the Neurosurgeons and was booked for repair of meningomyelocoele at the main theatre of the University of Port Harcourt Teaching Hospital, Rivers State, Nigeria. After medical stabilization and control of sepsis, the patient was scheduled for repair of the defect. The results of investigations done a week to the surgery were: Packed cell volume (%)- 42 (normal range: 40-54), Haemoglobin (gm/dl)= 14 (normal range: 15-18), WBC ($x10^9$/L), Neutrophils (%)- 86 (normal range: 70-85), Lymphocytes (%)- 34 (normal range: 20-45), Monocytes (%)- 8 (normal range: 2-10), Platelets ($x10^9$/L)-320 (normal range: 100-400). Serum bicarbonate (mmol/l) 20 (normal range: 24-30), creatinine (µmol/l)-25 (normal range 60-120), serum urea/sodium/potassium/sodium- within normal range. Liver function test- normal, blood culture-yielded no bacterial growth.

ANAESTHETIC MANAGEMENT

At preoperative review, history and laboratory investigation results were as already documented. Physical examination revealed an active neonate weighing 3.1 kg and in no obvious distress. She was not pale, afebrile, anicteric and not dehydrated. The anterior fontanelle was normotensive while the head circumference was 38.0 cm. Her respiratory rate was 38 breaths per minute, air entry was adequate and equal bilaterally, breath sounds were vesicular and there were no added sounds. Heart rate was 146 beats per minute and heart sounds were normal and no cardiac mumur was heard. The anaesthetic technique was explained to the baby's parents and they were reassured. An informed consent was then obtained from the neonate's father. A preoperative fasting instruction of no breast milk four hours to scheduled surgery was also discussed with the parents and documented in the patient' case file. She was categorised ASA class 2. A request was made for a unit of blood to be screened, grouped and cross-matched for possible intraoperative use.

Prior to the arrival in the operating theatre, routine check was carried out on the anaesthetic machine, monitors and suctioning machine. All the doses of anaesthetic drugs were calculated based on patient's weight, blood volume, allowable blood loss and fluid requirement were all calculated and written clearly on the protocol paper. The drugs were drawn up in labelled syringes. The resuscitation tray was set with intubation aids, emergency drugs, size 1 Macintosh and Wiscosin laryngoscope blades and plain portex endotracheal tubes of 2.5, 3.0 and 3.5mm internal diameter were selected. An introducer was placed in the lumen of the orotracheal tube most likely to be used (3.0mm). The assistance of a second anaesthetist and an experienced technician was obtained. The air conditioners in the operating room were turned off.

The patient was transferred from SCBU to the theatre in the prone position with 4.3% dextrose in 0.9% saline infused at the rate of 20 drops per minute using a soluset. Monitors were applied including precordial stethoscope and heart rate was 140 beats per minute. Arterial oxygen saturation was 98%, axillary temperature was 37^{o}C and ECG showed normal tracing. Operation room towels were folded into a ring and covered with sterile drape and the baby was turned to the supine position with the defect resting in the pocket of the ring. The radiant heater was positioned over the operating table.

Intravenous atropine (0.06 mg) was administered as premedication followed by preoxygenation with 100% oxygen by facemask at 4 litres per minute for 5 minutes. Anaesthesia was induced with halothane at increments of 0.5% after every 4 breaths to 3% concentration using the Jackson Rees modification of the Ayre's T-piece (MaplesonF) breathing system. When adequate depth of anaesthesia was reached, patient was assisted for about 2 minutes before laryngoscopy and tracheal intubation. Manual intermittent positive pressure ventilation was instituted using the Mapleson F breathing system and correct placement of the tube was confirmed by bilateral chest expansion and auscultation of air entry in both lungs as well as normal tracing on the capnograph. Halothane was then reduced to 0.5% and the oro-tracheal tube was then secured with adhesive tape. The pharynx was packed with moist gauze bandage.

Muscle relaxation was maintained with 0.5 mg of intravenous atracurium, with 2 top-up doses of 0.25 mg each. Analgesia was supplemented by intravenous pentazocine 2.5 mg and intravenous acetaminophen 150 mg. intravenous ceftriazone (250 mg) was administered as perioperative antibiotic. Surgery lasted for a duration of 1 hour 30 minutes. Intraoperative vital signs were a heart rate of 140-150 beats per minute, $Sp0_2$ of 98-100%, core temperature of 36.8- 37.2^{o}C, end tidal carbondioxide of 3.5-4.8 kPa and ECG which showed normal sinus rhythm. Estimated blood loss was 45mls and this was replaced with 20mls of whole blood. A total of 40mls of 4.3 dextrose saline was infused intraoperatively and urine output measured 15mls.

At the end of the operation, halothane was turned off and the lungs were ventilated with 100% oxygen for over 5 minutes. The pharyngeal pack was removed and the oropharynx was cleared of secretions with a sterile catheter. The residual neuromuscular blockade was reversed with 0.12 mg of neostigmine and 0.06 mg of atropine. There was full recovery from anaesthesia as evidenced by satisfactory respiratory efforts and arterial oxygen saturation of 100% on room air. The patient was extubated fully awake in a lateral position. She was transferred to the recovery room with precordial stethoscope in-situ to monitor her heart rate and air entry. Vital signs were monitored every 5minutes in the

recovery room. She was discharged to the SCBU in a satisfactory condition after 45 minutes.

SURGICAL DETAILS

With the patient in the prone position, an elliptical incision between a transition and normal skin was made. Incision was freed down to the extradural space. Nerve tissues were dissected away from the plaque. The dura was mobilized and closed over the plague with chromic catgut 3.0. The skin was closed with interrupted nylon 3.0 sutures and dressing with sterile gauze.

POSTOPERATIVE MANAGEMENT

The patient was nursed in the SCBU in a thermoneutral environment in a prone position. Maintenance fluid was 340mls of 4.3% dextrose in 0.18% saline over the first 24 hours. Breast milk was commenced on the 1st postoperative day and was well tolerated. Urine output was 150mls in the 1st 24 hours. Postoperative antibiotic included ceftriaxone 150mg daily and gentamicin 15mg daily. Postoperative analgesia was achieved with IV acetaminophen 50 mg 6hourly for 48-hours and thereafter with syrup acetominophen 5mls 4 hourly for 5 days. She also received syrup vitamin C 8-hourly. Postoperative haemoglobin was 13.8 g/dl. There was progressive improvement, although her incontinence persisted and she was discharged to be reviewed at out-patient clinic on the 20th postoperative day.

DISCUSSION

Defects of the neural tube are induced by damage occurring during the fourth week of fetal development and could result from a combination of genetic and environmental factors.[2,8]The majority of the defects of the spinal neural tube occur in the lumbo-sacral region.[2] In spina bifida there is absence of the vertebral arches and failure of closure of the overlying skin.[9]Spina bifida cystica can be further subdivided into meningomyelocoele and meningocoele. In meningocoele(which accounts for 20% of spina bifida cystica) the spinal cord is normally formed but the thin sac of meninges which contains cerebrospinal fluid (CSF) is exposed. While Myelomeningocoele(representing 80% of cases of spina bifida cystica)manifests with a child that is born with meningocoele (which may rupture at birth) and abnormally formed spinal cord.[2]Meningomyelocoele is usually associated with sensory and motor deficit that depends on the level of the lesion. The anal sphincter may be patulous and if paralysed, there may be a distended and neurogenic bladder with resultant faecal and urinary incontinence.[10] Corrective surgery is planned within few days of birth. If surgery is delayed, the sac grows out of proportion to the growth of the child and the overlying skin becomes atrophic and ulcerates.

Prenatal diagnosis of neural tube defects is suggested by presence of raised alpha-feto-protein levels in blood and amniotic fluid.[11] Ultrasound examination of the head may detect early hydrocephalus which occurs in about 90% of children with meningomyelocoele.[11]And, ultrasound examination of the kidneys may reveal hydronephrosis secondary to bladder paralysis[11]. Poor prognosis of meningomyelocoelemight be indicated by a large thoracolumbar lesion, scoliosis or kyphosis birth; gross hydrocephalus and other severe congenital anomalies. Children with these featuresrecord minimal benefit from surgical treatment.[2,9] The patient did not have any of these severe abnormalites and hence was booked for surgery.

Detailed preparation plays a major role in the successful outcome of neonatal anaesthesia and surgery thus management of the neonate in the SCBU provides the best level of neonatal care.[9]Results of relevant investigations are reviewed and actioned upon before surgery to minimise untoward intra-operative event. Preoperative preparations should include establishment of an intravenous access, maintenance of the airway, adequate oxygenation, normothermia and correction of acidosis, dehydration, hypovolaema and hypoglycaemia.

Meningomyelocoele may be associated with some renal abnormalities, and preoperative review of the renal status is essential. Serum electrolyte, urea and creatinine estimation may reveal electrolyte imbalances as well as sepsis. Electrolyte imbalance is associated with delayed emergence following muscle relaxation with non-depolarising muscle relaxants and should be corrected before administration of anaesthesia. Preoperative haemoglobin should be estimated, and any pre-existing anaemia corrected in order to improve oxygen carrying capacity of the blood, and tissue oxygenation. Blood loss is poorly tolerated by neonates and transfusion is required if up to 10% of the total blood volume is lost.[4] The total blood volume in a neonate is about 85mls/kg and 90mls/kg in preterm babies. Grouping and cross-match of blood for possible intraoperative blood transfusion is often indicated.

Patients with meningomyelocoele are prone to evaporative fluid loss. Leakage of cerebrospinal fluid (CSF) from ruptured meningomyloecele may result in electrolyte imbalance and dehydration. The loss of protective covering also increases the risk of infection of the central nervous system. A search for neonatal sepsis, meningitis and acidosis is often indicated in these neonates. Sepsis is one of the causes of neonatal jaundice and may be noticed within the 1st 24 hours of life. Antibiotic prophylaxis and coverage of the lesion with sterile gauze soaked with normal saline to prevent fluid loss and infection are usually indicated and preoperative fluid replacement is necessary. To compensate for the continuous fluid loss from ruptured meningomyelocoele and prevent dehydration, adequate preoperative fluid replacement was ensured by administration of 240mls of daily maintenance fluid in addition to the 3-hourly breast-feeding.

Preoperative fast reduces the risk of regurgitation and aspiration of gastric content but exposes neonates to hypoglycaemia if prolonged. This could occur because neonates have relatively high metabolic rate, lower glycogen stores, and immature mechanism of gluconeogenesis compared to older children.[7] Neonates also have larger surface area to weight ratio than adults which increases fluid loss from evaporation.[7] In order to prevent dehydration and hypoglycaemia, unlimited amounts of clear fluids and breast milk may be allowed up to 2 and 4 hours respectively prior to induction of anaesthesia.[7] No significant difference has

been observed in intra-gastric volume and PH of children who are allowed to ingest clear fluids up to 2-3 hours when compared to children who are denied clear fluid 2-3 hours prior to induction of anaesthesia.[6,7]Normoglycaemia could be successfully maintained by infusion of 10% dextrose in lactated Ringer's solution at 3-4mls/kg/hour.

Sedative premedication is usually not indicated in neonates. Atropine premedication should be administered to dry excessive secretion and to prevent bradycardia associated with administration of suxamethonium, use of volatile agents and intraoperative hypoxia in children.[6] The sympathetic nervous system is less developed in the neonate and results in dominance of para-sympathetic (vagal) tone. Cardiac output is dependent on heart rate in the neonates.[7] Neonates have immature cardiac muscles and cannot increase stroke volume to maintain cardiac output.Infants have a very poor central thermoregulation, thin insulating fat, increased body surface area to mass ratio and high minute ventilation.[7] Therefore are more likely to develop hypothermia under anaesthesia. Hypothermia has been associated with delayed awakening from anaesthesia, myocardial ischaemia, respiratory depression, increased pulmonary vascular resistance, altered drug responses and increased postoperative mortality.[12] The neonate increases metabolism of highly perfused interscapular and retroperitoneal fat (brown fat) in order to maintain the core temperature if exposed to hypothermic conditions. This endogenous heat production is referred to as non-shivering thermogenesis and is associated with increase in oxygen consumption. Thermal stress elevates plasma catecholamine levels, causing pulmonary and systemic vasoconstriction and may result in reversal to fetal circulatory pattern.[13]

Use of over-head radiant heater provides adequate warmth to the patient and makes it possible for the theatre temperature to be kept at a level comfortable for the operating room personnel. Also, use of over-head radiant warmer during induction and emergence, placing the intubated patients head and limbs in plastic sandwich bags and covering the remainder of the patient with an adhesive plastic drape or reflective blanket minimize loss of heat to the environment.[14] Hypothermia can also be prevented by maintaining a warm operating room, warming all intravenous fluids, humidification of inspired gases, warming blanket and warming lights.[15,16] Other heating devices include heated mattresses and fan heater. The risk of thermal injuries from these heating devices is prevented by keeping the radiant heater at a distance from the skin. The working temperature of heating devices should also not exceed $40^{o}C$.[13]

Hypoxia is a major cause of preoperative morbidity and mortality in paediatric patients. The neonates have smaller functional residual capacity in relation to oxygen consumption. This results in earlier onset of hypoxia during periods of apnoea such as during laryngoscopy. The presence of fetal haemoglobin in the circulation which has higher affinity for oxygen reduces the release of oxygen to the tissues. Reversal to fetal circulation with worsening hypoxia can occur if neonates are exposed to hypoxic conditions. Pre-oxygenation with 100% oxygen for 5 minutes before the induction of anaesthesia is therefore mandatory in order to increase alveolar oxygen concentration, thus reducing the risk of hypoxia during this period of apnoea.

The technique for induction of anaesthesia depends on several factors including postnatal and post-conceptual age of the neonate, physical status, relative risk of pulmonary aspiration of regurgitated gastric content and nature of the surgical procedure.[7] The goals are to maintain cardiopulmonary stability while permitting elimination of pain perception and major stress response and to secure airway and control ventilation of the lungs as quickly as possible. Inhalational induction is favoured if there is no risk of aspiration and if difficult intubation is anticipated. Inhalational induction of anaesthesia is usually easy in the neonates and small children due to high minute volume ventilation and low functional residual capacity. Halothane has a pleasant smell and is not irritable to the airway, so it is an excellent induction agent for neonates and children.[17] The healthy neonates and young infants have higher susceptibility to cardiovascular depression from halothane than older children.[18] Clinically significant arterial hypotension and bradycardia occurs in 50% of infants anaesthetised with halothane.[19] This is because the uptake of nitrous oxide and volatile anaesthetic agents and their distribution to vital organs are more rapid.[19] Both halothane and isoflurane in concentrations below one minimum alveolar concentration (MAC), if supplemented by nitrous oxide or narcotics provides adequate anaesthesia and circulatory stability in all but the very ill and very small neonates.[20] Isoflurane is irritant to the airwayand therefore not popular for induction of anaesthesia.[21]Sevoflurane is preferable if available as it provides smooth and rapid induction profile in infants.[22]

Intravenous induction of anaesthesia is rapid and preferable where there is risk of aspiration of gastric contents. Thiopentone and ketamine offer practical means of achieving rapid induction of anaesthesia for the purpose of tracheal intubation in the neonate, with the same secondary effect found in older patients. But, ketamine is preferred because of minimal circulatory depression compared to thiopentone.[23]Suxamethonium rapidly produces excellent muscle relaxation for tracheal intubation. However, neonates might be resistant to suxamethoniumand doses up to 2-3mg/kg are often required unlike adults in whom dose of 1mg/1kg is usually sufficient.[7,20] Children are more prone to cardiac dysrhythmias, myoglobinaemia and hyperkalaemia than adults. The risk of malignant hyperthermia following its use is higher in the paediatric age group.[7] For these reasons, succinylcholine is best avoided for routine elective surgery in children. Rocuronium, a short acting non-depolarizing muscle relaxant is devoid of these side effects and if available, presents an ideal alternative to suxamethonium.[24]

The anatomy of the neonate makes mask ventilation and intubation difficult.[25] They have a larger head relative to the body, larger tongue, narrow nasal passages, anterior and cephalad larynx (at a vertebral level of C4 versus C6 in adult), U-shaped floppy epiglottis, a short trachea and neck.[25] They also have prominent occiput which tends to place the head in a flexed position prior to intubation.

Maintaining the neonate's head in a correct position during mask ventilation and laryngoscopy reduces soft tissue airway obstruction and enhances visualization of the larynx. The optimum position for intubation involves moderate flexion of the cervical spine and extension of the atlanto-occipital joint. In the supine position with the shoulders flat on the operating table, the neonate's prominent occiput causes the appropriate cervical flexion and, elevation of the symphysis menti results in the desired extension of the atlanto-occipital joint.[20]

Intubation is easier with the aid of a Magill's laryngoscope with the tip of laryngoscope blade placed behind the epiglottis instead of the vallecula in the adult patient. The use of Miller's laryngoscope equipped with canula and proximal nipple attachment for continuous oxygen administration during laryngoscopy could reduce hypoxia in infants during tracheal intubation.[26] The stress of laryngoscopy and tracheal intubation could be deleterious in the neonate as this mightcause intra-cranial haemorrhage therefore, adequate anaesthesia should be ensured.The cricoid cartilage is the narrowest point of the airway in children younger than 5 years of age unlike adults. An endotracheal tube that passes through the glottis could still impact on the cricoid cartilage, causing ischaemia, post-operative largryngeal oedema and stridor. One milimetre of oedema will have a proportionately greater effect in children because of their smaller tracheal diameters. Therefore, endotracheal tube with internal diameter of 3.0mm is usually adequate for term neonates[7] was used. The reinforced tube would have been preferred to avoid kinking in abnormal position if it were available.

Regardless of the method of induction of anaesthesia, ventilation of the lungs with inspired oxygen concentration of 100% for 2 minutes before and after induction reduces hypoxaemia associated with apnoea during intubation.[7] The narrow airway in neonates increases airway resistance, thus increasing the work of breathing. Controlled lungs ventilation is often preferred, especially in intubated patients. Mapleson-F breathing system is valveless, provides low resistance to flow of gases and reduces the work of breathing during spontaneous respiration. A minimum of two hundred and fifty percent of the minute volume is required to prevent re-breathing.

Compression of the abdomen may occur in the prone position resulting in pooling of blood in the extremities thereby decreasing preload, cardiac output and blood pressure. Also, compression of the abdomen and thorax will decrease total lung compliance and increase the work of breathing. Care should therefore be taken to elevate the chest and pelvis with soft pads in such a way that abdominal movement is unhindered. The slight lateral rotation of the head while in prone position avoids kinking of the orotracheal tube and also prevents pressure on the baby's nose. But, extreme rotation may cause decrease in cerebral venous drainage and blood flow.[25]

The response of neonates to non-depolarizing muscle relaxant is variable. The neonate has greater volume of distribution combined with hepatic and renal immaturity resulting in a slower rate of metabolism and excretion.[25,27] This prolongs the effect of non-depolarizing muscle relaxant that are subject to hepatic metabolism especiallypancuronium, vecuronium and to a lesser extent rocuronium. Atracurium was considered the ideal relaxant because it relies on Hofmann's elimination and ester hydrolysis (PH and temperature dependent) for excretion and its intermediate action is desirable for immediate post-operative extubation.[28]

The high metabolic demand and high body surface area to weight ratio in children is considered in the calculation of their fluid therapy. This forms the basis for calculating maintenance fluids as suggested by Haliday and Segar.[29]There was no fluid deficit from pre-operative fasting in this child because she was on maintenance intravenous fluids. The intraoperative fluid management in this baby thus considered maintenance requirement of 4mls/kg/hour for $1^{1/2}$ hours duration of anaesthesia, third space loss and blood loss. The third space loss and blood loss were difficult to measure because they were mixed up. However, because of the continuous leakage of CSF, third space loss was estimated as 5ml/kg/hour and a total of 40ml of fluid was infused over the 90minutes duration of anaesthesia. The risk of fluid over load is higher in neonates and this risk could be reduced by use of soluset which delivers smaller, regulated volumes of infusion than conventional infusion giving sets. Programmable infusion devices are more accurate.

The composition of intravenous fluid is important in the neonate. Routine use of glucose containing infusion for short procedures has been questioned because of increased incidence of hypoxic brain damage observed in animals with high blood glucose concentration.[30] However, concerns about unrecognized hypoglycaemia has motivated the routine use of glucose containing infusion in children; particularly those who have not had any food or drinks for longer than usual time or those who have diminished glucose stores.[31]

The repair of meningomyelocoele might be associated with significant blood loss, much of which could be concealed by drapes. The acceptable pre-operative haematocrit is 35%.[20] Allowable blood loss (starting haematocrit minus allowable haematocrit divided by current haematocrit and multiplied by estimated blood volume) less than 10mls/kg should be replaced with Ringer's lactate solution but blood loss more than 10ml/kg should be replaced with colloids.[20]Intra-operative access to paediatric patients after surgical draping is usually limited because of their small size therefore meticulous use of monitors is mandatory. Precordial stethoscope should be applied on the back of the prone patient for continuous monitoring of breath sounds and early detection of displaced endotracheal tube. Monitoring of arterial oxygen saturation and end tidal carbon dioxide is also necessary in order to ensure adequate lung ventilation and tissue oxygenation. Bright red blood is an indication of adequate oxygenation and the colour of the blood at the operation field should be monitored. A dry operation field might indicate hypoperfusion or cardiac arrest.

The adequacy of reversal of neuromuscular blockade should be assessed by peripheral nerve stimulator before the patient is extubated. In the absence of a peripheral nerve stimulator,

it can be assessed clinically. The presence of flexion of the hips with raising of one leg or both off the operating table has been found to correspond to respiratory force of -35 cmH_20 which is a reliable indicator of adequate reversal of neuromuscular blockade in children[32]. The combination of this method and observation of satisfactory respiratory efforts and normal arterial oxygen saturation during spontaneous respiration can be used to assess the adequacy of reversal before the patient is extubated.

The management of pain in children is often inadequate and there is no evidence to support the idea that pain is less intense in neonates and young children due to their developing nervous system. Adequate analgesia is essential for good outcome in neonates undergoing surgery.[33] A regular schedule of postoperative analgesia with acetaminophen and pentazocine is often necessary to ensure continuous analgesia since the neonate cannot complain of pain. Opioid analgesics readily cross the less developed blood-brain barrier of the neonates and may cause postoperative respiratory depression.[34] Opioids should therefore be administered with caution. Remifentanil, a short acting opioid depends on tissue and plasma esterases for metabolism and may be preferred. Neonates who receive intraoperative opioid should be monitored for postoperative respiratory depression.[34] Narcotic antagonists are effective immediately after administration but have relatively short duration of action after a single dose and might therefore require repeated doses.

CONCLUSION

Anaesthesia for repair of meningomyelocoele in a neonate poses a unique and formidable challenge to even anaesthetists. Neonates, in addition to the basic physiologic, anatomic and pharmacologic differences compared to older children are prone to illnesses that require unique surgical and anaesthetic strategies. Proper understanding of these peculiar differences, well-managed anaesthesia and surgery, consistent monitoring and attention to details are required for a favourable outcome.

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Conflict of interest: None

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